REQUEST FOR RECONSIDERATION

Restriction has been required from between:

1) Group I, claims 5-19, drawn to methods of administering antibodies that bind FcαR1; and
2) Group II, claims 20-25, drawn to antibodies that bind FcαR1.

As noted above, Applicants provisionally elect with traverse to prosecute claims 5-19 (Group I) Applicants also provisionally elect the species allergy, and, in particular, asthma.

Restriction is said to be justified inasmuch as, pursuant to PCT Rules 13.1 and 13.2, no 'single general inventive concept' is presented, and these claim group 'lack the same or corresponding special technical features', respectively. The basis for these conclusions is said to be WO 02/064634.

However, is it noted that claim 6 of the present application recites that 'the monovalent antibody fragments are directed against EC2 domain of the FcαR1 receptor.

In contrast, WO 02/064634 discloses a monoclonal antibody that binds to CD89 at a site which is at or near the IgA binding site, although the antibody does trigger at least one Fc receptor-mediated effector cell activity. This cited reference clearly neither discloses nor suggests monovalent antibody fragments directed against EC2 domain of the FcαR1 receptor.

Clearly, the present claims pertain to subject matter that define a contribution over WO 02/064634. Hence, the two claim groupings satisfy both PCT Rules 13.1 and 13.2.

Accordingly, it is concluded that the 'unity of invention' standard has been satisfied. Examination of both claim groupings should now proceed without further delay.

Furthermore, although Applicants have provisionally elected the species allergy, and, in particular, asthma, this is with traverse.

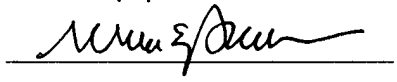
Notably, the claimed method of treating inflammatory disease of Group (I) is not limited to treating allergy or, in particular, asthma; but it rather intended as a treatment for inflammatory diseases generally. It is noted from page 5, lines 21-24, of the application as filed that:

The anti-inflammatory properties of said monovalent antibody fragment result from a down-regulation of the pathological inflammatory reactions involving FcαR1 – expressing myeloid cells.

Hence, a reasonably complete search of inflammatory disease treatment should be conducted.

Favorable reconsideration is earnestly solicited.

Respectfully submitted
THE NATH LAW GROUP



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